

# United States Air Force Research Laboratory



**THE IMPACT OF HIGH LEVELS OF NITROGEN IN THE  
BREATHING GAS AND IN-FLIGHT DENITROGENATION  
ON THE RISK OF DECOMPRESSION SICKNESS (DCS)  
DURING SIMULATED ALTITUDE EXPOSURE**

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## **INTRODUCTION**

The research described in this Technical Report was requested and funded by the US Special Operations Command (USSOCOM) in response to Air Force Special Operations Command (AFSOC) flight safety concerns. Three specific issues were addressed and this report is divided into three sections accordingly.

**Phase I: Is there an increased risk of altitude decompression sickness (DCS) resulting from the use of the NORMAL setting (oxygen/nitrogen mixture) on the Narrow Panel Regulators of the AC-130 SPECTRE Gunships when compared to the 100% oxygen setting? If so, how much greater is that risk?**

**Phase II: Will high levels of nitrogen in the breathing gas mixture produced by the On Board Oxygen Generating System (OBOGS) of the new AFSOC CV-22 Osprey result in an increased risk of DCS when compared to breathing 100% oxygen? If so, how much higher will that risk be?**

**Phase III: The CV-22 is expected to operate unpressurized up to altitudes of 25,000 ft for long periods of time. A very high risk of DCS is predicted for such operations. Assuming the use of ground level preoxygenation (prebreathing) for reducing DCS risk is impractical for AFSOC, how effective would in-flight (stage) denitrogenation by breathing 100% or OBOGS product gas be for reducing DCS risk?**

Altitude DCS can occur when personnel are decompressed to a combination of altitudes and exposure times sufficient to elicit evolved gas (mostly nitrogen) in the tissues (9). Bubble formation in the body can result in symptoms ranging from mild pain to serious neurological/respiratory impairment. For exposures above 18,000 ft the primary countermeasure is preoxygenation (prebreathing). This procedure consists of breathing 100% oxygen for some period of time prior to and during ascent to altitude. By breathing 100% O<sub>2</sub> there is zero nitrogen in the inspired breathing gas, creating the maximum driving force for nitrogen elimination from the body (denitrogenation). Continuing to breathe 100% O<sub>2</sub> during the altitude exposure further reduces the N<sub>2</sub> in the tissues, as well as preventing hypoxia.

The rate of denitrogenation varies primarily with altitude, time of exposure, prebreathe time, and changes in circulation (e.g., exercise). The differential set up by these variables between the partial pressure<sup>1</sup> of N<sub>2</sub> in the tissues and the partial pressure in the ambient breathing gas can be referred to as *the nitrogen partial pressure gradient or the PN<sub>2</sub> gradient*. Thus, another variable often said to define the PN<sub>2</sub> gradient is the breathing gas mixture. As mentioned above, the exclusion of nitrogen in the breathing gas, i.e. prebreathing with 100% oxygen, maximizes the PN<sub>2</sub> gradient. Therefore, it is generally accepted that the more N<sub>2</sub> in the breathing gas, the less effective the denitrogenation and the higher the risk of DCS.

However, there is only limited evidence in the altitude physiology literature to support this concept.

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<sup>1</sup> In a mixture of gases such as air, the proportion of the total pressure contributed by a single gas in the mixture is called *partial pressure*.

Barer et al. (1) found that greater than 10% N<sub>2</sub> during prebreathing at ground level nullified the denitrogenation effect. An AFRL study on the effects of breathing a 50% nitrogen/50% oxygen mixture at altitude showed significantly more circulating venous gas emboli while breathing the 50/50 mix than with the use of 100% O<sub>2</sub> (17). Unfortunately, that study was done at low altitudes, 16,500 ft and below, and measured only circulating gas emboli, since DCS did not occur. It has been found that circulating gas emboli are not good predictors of the onset of DCS symptoms (10). Work at higher altitudes with mixed-gas breathing has not been documented, and, therefore, the effect of high N<sub>2</sub> breathing mixtures on the DCS risk at altitude is not well understood.

Oxygen systems in military aircraft using the USAF Narrow Panel Regulator have two regulator settings: 100% O<sub>2</sub> and NORMAL. The NORMAL setting dilutes the air at the lower altitudes with O<sub>2</sub>, thereby reducing consumption of aircraft O<sub>2</sub> stores and reducing the potential for pulmonary acceleration atelectasis. Figure 1 shows the minimum percent oxygen in the breathing gas with changing altitude. At 18,000 ft, the minimum O<sub>2</sub> concentration is approximately 38% when using this system. At 25,000 ft, the minimum O<sub>2</sub> concentration is 52% and reaches 100% at approximately 33,000 ft. Thus, it is of interest to determine if these high levels of N<sub>2</sub> in the breathing gas significantly contribute to DCS risk, particularly in unpressurized aircraft.

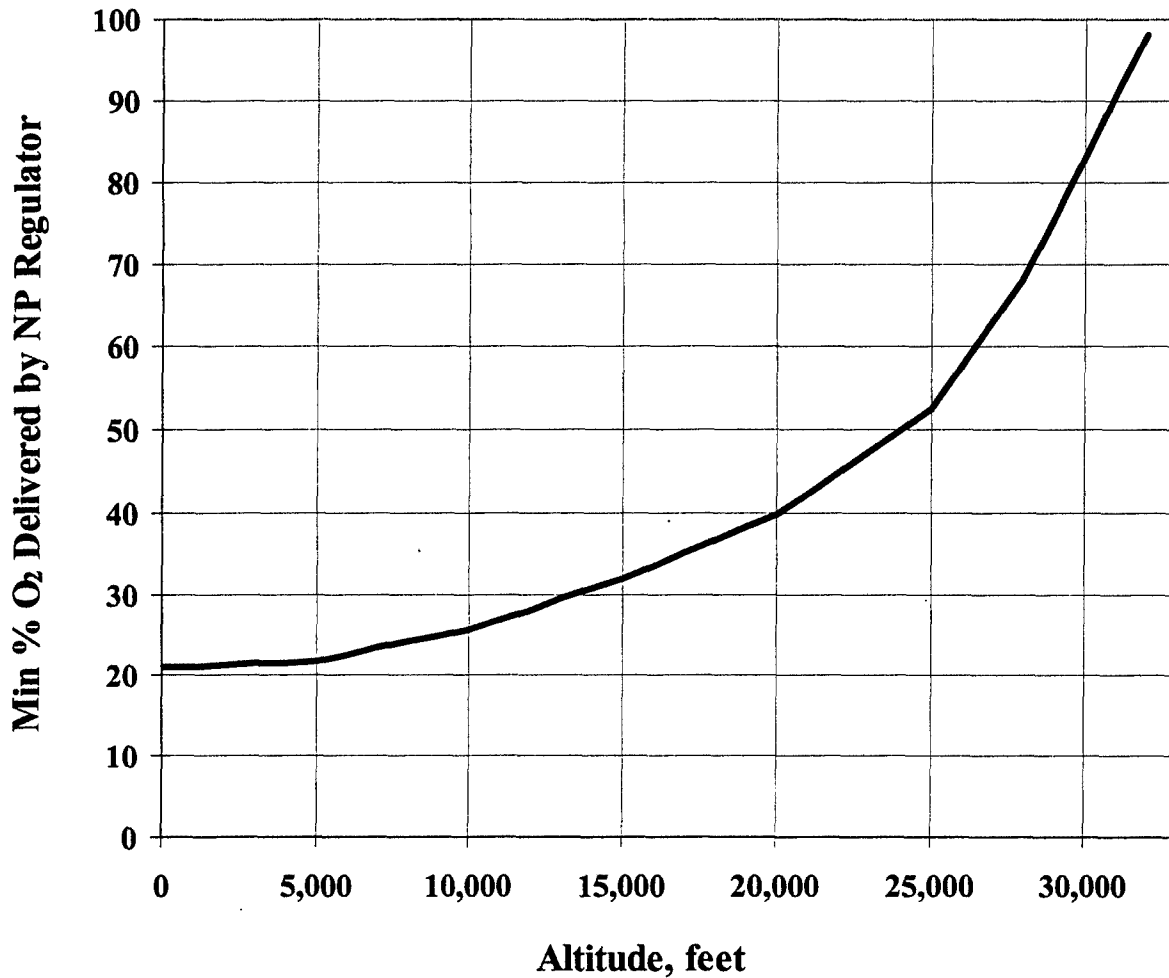


Figure 1. Minimum concentration of oxygen (%) delivered by USAF Narrow Panel Regulator (MIL-R-83178; USAF)

Currently, most military aircraft are replacing the liquid oxygen and high pressure gas systems with On Board Oxygen Generating Systems (OBOGS). This molecular sieve oxygen concentrator technology is used for generating oxygen-enriched breathing gas to prevent hypoxia in unpressurized aircraft and pressurized high-altitude aircraft. OBOGS does not generate 100% O<sub>2</sub>. A number of parameters such as altitude, number of people breathing on the system, supply of air, temperature, and others determine the exact make-up of the OBOGS product gas. Under some conditions, the N<sub>2</sub> concentration can reach as high as 40% at FL180. Under the “best”

conditions, OBOGS product gas concentrations running near optimal performance can be 93.0% O<sub>2</sub>, 4.2% argon, and 2.8% N<sub>2</sub>. This is the highest O<sub>2</sub> concentration OBOGS can deliver. The example in Figure 2 shows the percent oxygen in the CV-22 OBOGS product gas. Since this aircraft operates with a crew of four, the highest O<sub>2</sub> level that can be produced under the conditions indicated is approximately 82%.

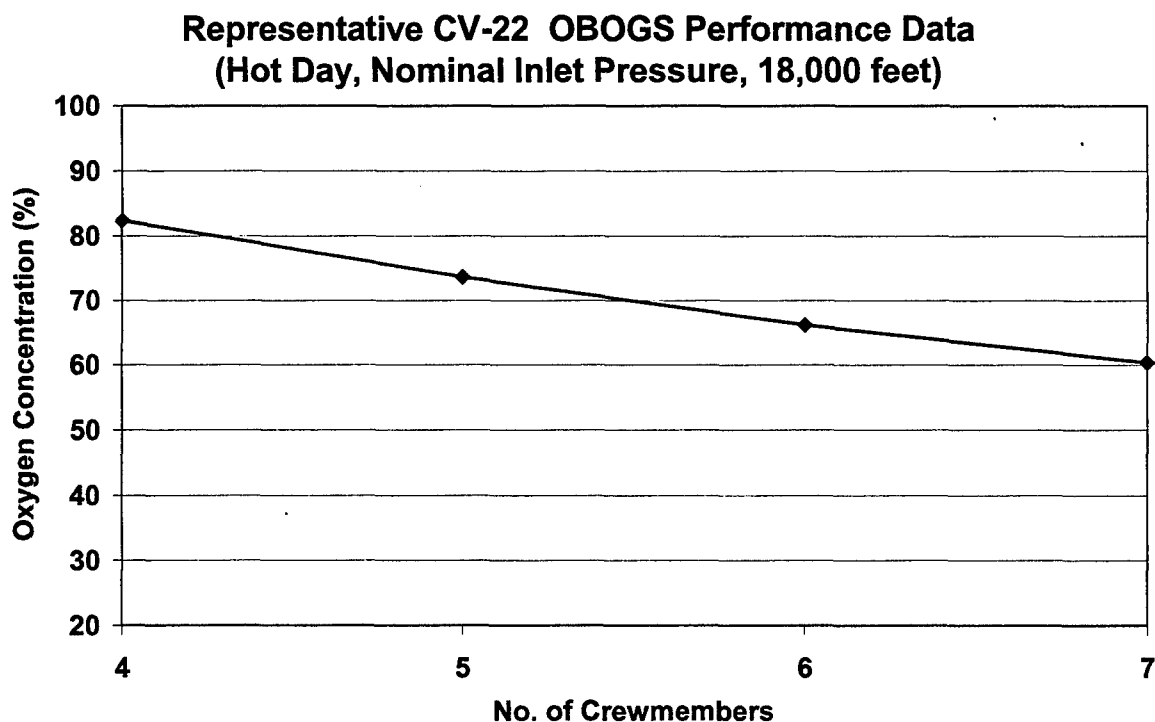


Figure 2. Example of CV-22 OBOGS oxygen concentration

As noted above, the OBOGS product gas also contains another inert gas, argon. Argon has lower diffusivity than nitrogen, but a higher solubility in fatty tissue. Thus, if the argon concentration is low and exposure time short, its impact on bubble growth and DCS risk should be minimal. Cooke et al. (3) studied the effect of argon on DCS but reported results that were inconclusive. A recent study in our lab found an increase in DCS when an argon/oxygen mixture

was breathed for 4 hours at 18,000 ft just prior to ascending to 35,000 ft for 3 hours (13).

However, the argon concentration in the breathing gas of that study was much higher (62%) than that found in OBOGS product gas. Thus, the impact of argon on DCS risk when using OBOGS is not known.

Aircraft oxygen systems deliver breathing gases with varying amounts of  $N_2$ , and it is generally assumed that breathing high levels of  $N_2$  will result in slower denitrogenation and a higher risk of DCS than breathing 100% oxygen. Data were needed to define the effect of increasing levels of  $N_2$  in the breathing gas on the DCS incidence during altitude exposures at 18,000 ft and above. Further, the impact of argon, albeit small, from OBOGS on the DCS risk also needed definition. As altitude increases (total ambient pressure decreases), the  $PN_2$  gradient from the tissues to the air being breathed by a crewmember will also increase. This study tested the hypothesis that the increasing  $PN_2$  gradient between the ground level tissue  $N_2$  saturation and the ambient  $PN_2$  at and above altitudes of 18,000 ft does not increase DCS risk while breathing high percentages of  $N_2$  when compared to the DCS risk found while breathing 100%  $O_2$ . In addition, we hypothesized that the small amount of argon from the OBOGS product gas would have no impact on the DCS risk.

If ground level preoxygenation cannot be used for practical reasons as a countermeasure for reducing DCS risk, breathing 100% (or OBOGS product gas) in the aircraft while enroute could serve as an alternative method of denitrogenation. This in-flight or stage denitrogenation has been shown to be as effective as ground level prebreathe when done at altitudes at or below 16,000 ft (21). This study compared the effectiveness of in-flight denitrogenation at 16,000 ft using both 100%  $O_2$  and OBOGS product gas on DCS risk reduction.

## METHODS

The voluntary, fully-informed consent of the subjects used in this research was obtained in accordance with AFI 40-402, and the protocol was approved by the AFRL Institutional Review Board at Brooks AFB and the USAF Surgeon General's Office. All subjects passed an appropriate physical examination and were representative of the USAF rated aircrew population. They were not allowed to participate in SCUBA diving, hyperbaric exposures, or flying for at least 48 hours before each scheduled altitude exposure. Prior to each altitude exposure, a physician conducted a short physical examination of subjects to identify any signs of illness or other problem that would endanger the subject or bias the experimental results. Chamber ascent and descent were at a rate not exceeding 5,000 fpm. A neck-seal respirator made by Intertechnique® (Plaisir Cedex, France) was used to deliver the breathing gas. This mask provided a slight (2 cm of water) positive pressure which reduced the opportunity for inboard leaks of air from the atmosphere and was more comfortable than the standard aviator's mask. At 15-min intervals, the subjects were monitored for venous gas emboli (VGE) using a Hewlett Packard® SONOS 1000 Doppler/Echo-Imaging System. This system permits both audio and visual monitoring and recording of gas emboli in all four chambers of the heart. VGE were graded using a modified Spencer Scale (16).

Mild exercise consisting of three upper-body exercises as described in Webb et al. (19) was performed by the subjects at intervals throughout the altitude exposure. The subjects walked less than ten steps between exercise stations and the echo-imaging station at 4-min intervals.

AFRL Medical Monitors insured subject health and safety, and made the diagnosis of DCS. These Medical Monitors were not investigators on the protocol in order to provide for unbiased

diagnosis. Subjects were alone in the chamber while at simulated altitude. The echo-imaging transducer was placed using a robotic arm operated from outside the chamber. The subjects were instructed to report any changes in well-being to the Medical Monitor and the determination to terminate the exposure was made from these reports. The subjects were examined after recompression to ground level. The Medical Monitors were trained in the diagnosis of DCS and had the ability to consult with the physicians in Hyperbaric Medicine within the same building. Endpoints of the exposures were: 1) completion of the scheduled exposure period, 2) diagnosis of DCS, or 3) detection of left ventricular gas emboli (LVGE). A more detailed description of the endpoints can be found elsewhere (14).

Subjects were not questioned about how they felt during the altitude exposures. To provide relief from boredom and more closely emulate operational distractions, movies were shown to the subjects during the hypobaric exposures. The subjects received a briefing on the morning of each exposure which emphasized their responsibility to report any DCS symptoms or change in well-being to chamber personnel, and a list of symptoms was posted in plain view inside the chamber.

The significance of the response, DCS or no DCS, of subjects was analyzed using the Chi Square test. Log Rank and Wilcoxon's tests were used to compare homogeneity of curves representing cumulative incidence of DCS and VGE versus time.

#### *Phase I exposure profiles:*

Two experimental altitude exposure profiles (Tests A and B) were used in this phase of the study to determine the effect of high levels of N<sub>2</sub> in the breathing gas on DCS risk while at altitude. The details of each profile are in Table I. Test A used 60% nitrogen (partial pressure N<sub>2</sub> at 228 mm Hg) and 40% oxygen as a breathing mixture at 18,000 ft. The subjects performed



mild exercise (20), had zero prebreathe, and were exposed for 4 hr (N=30). Test B was identical to Test A, except heavy exercise (dual cycle ergometry at 50% VO<sub>2</sub> peak) was used at altitude (N=29). The controls for these two tests using 100% O<sub>2</sub> for a breathing gas had previously been accomplished in our lab and the data were available in the AFRL DCS Research Database.

**Table I: Phase I experimental exposures**

	Test A	Test B
# of subjects in tests	30	29
# of subjects in controls	20	30
Altitude (ft)	18,000	18,000
PN <sub>2</sub> (mmHg)	228	228
N <sub>2</sub> gradient (mmHg)	355	355
Gas mixture	60%N <sub>2</sub> /40% O <sub>2</sub>	60%N <sub>2</sub> /40% O <sub>2</sub>
Prebreathe (min)	N/A	N/A
Activity	mild exercise	heavy exercise

*Phase II exposure profiles:*

Test C was at 22,500 ft with mild exercise (20), zero prebreathe and 4-hr duration (N=40). Test D was used to determine the effect of argon in the breathing gas on DCS risk. Test D consisted of 90 min of preoxygenation followed by a 240-min exposure to 25,000 ft. The breathing gas used during both the preoxygenation period and during the altitude exposure was 93% oxygen, 4.2% argon, and 2.8% nitrogen, representing the highest oxygen level generated by most OBOGS systems. This Test D included 40 subjects. The control for Test C using 100% O<sub>2</sub> for a breathing gas had previously been accomplished in our lab and the data were available in the AFRL DCS Research Database.

**Table II: Phase II experimental exposures**

	Test C	Test D
# of subjects in tests	40	40
# of subjects in controls	40	ADRAC
Altitude (ft)	22,500	25,000
PN <sub>2</sub> (mmHg)	126	8
N <sub>2</sub> gradient (mmHg)	457	575
Gas mixture	40% N <sub>2</sub> /60% O <sub>2</sub>	2.8%N <sub>2</sub> /4.2%Ar/93%O <sub>2</sub>
Prebreathe (min)	N/A	90
Activity	mild exercise	mild exercise

However, no such control for Test D was available in the database. Therefore, the control for Test D was generated by a DCS risk prediction model developed at AFRL. This model is the basis for the Altitude DCS Risk Assessment Computer (ADRAC) (8). This model, based on the loglogistic distribution, is used to predict the probability/risk of DCS over time as a function of altitude, preoxygenation time, exposure time, exercise, and the time of onset of maximum venous gas emboli grade (4,5). A prospective series of human trials successfully validated the predictive ability and accuracy of this model (6,15). Detailed descriptions of the ADRAC model can be found elsewhere (12, 15).

#### *Phase III exposure profiles:*

The two exposure profiles in Phase III were identical except for the breathing gas. In Test E the subjects breathed 50%N<sub>2</sub>/50%O<sub>2</sub> while in Test F they breathed 100% O<sub>2</sub>. There was no ground-level prebreathing. The subjects breathed air during the ascent until they reached 10,000 ft. Operationally, supplemental oxygen is not used until 10,000 ft is reached. At 10,000 ft, the subjects put on the masks and breathed either the 50/50 mix or 100% O<sub>2</sub> for the rest of the exposure. In either exposure they stayed at 16,000 for 3 hours before ascending to 25,000 ft, where they remained for 4 hours. Both ascents were done at 1,000 ft/min. At 16,000 ft they were at rest, while at 25,000 ft they performed mild exercise as described. The nitrogen partial

pressure gradient at 16,000 ft was 377 mm Hg, while at 25,000 ft it increased to 442 mm Hg when using the 50/50 mix (Test E). As with all of the exposures, when using 100% O<sub>2</sub> the PN<sub>2</sub> gradient was 583 mm Hg throughout the exposures. There were 40 subject-exposures completed for each test. Test F was considered the control, and both tests were compared to the results of previous AFRL DCS Database exposures involving no prebreathe and no in-flight denitrogenation.

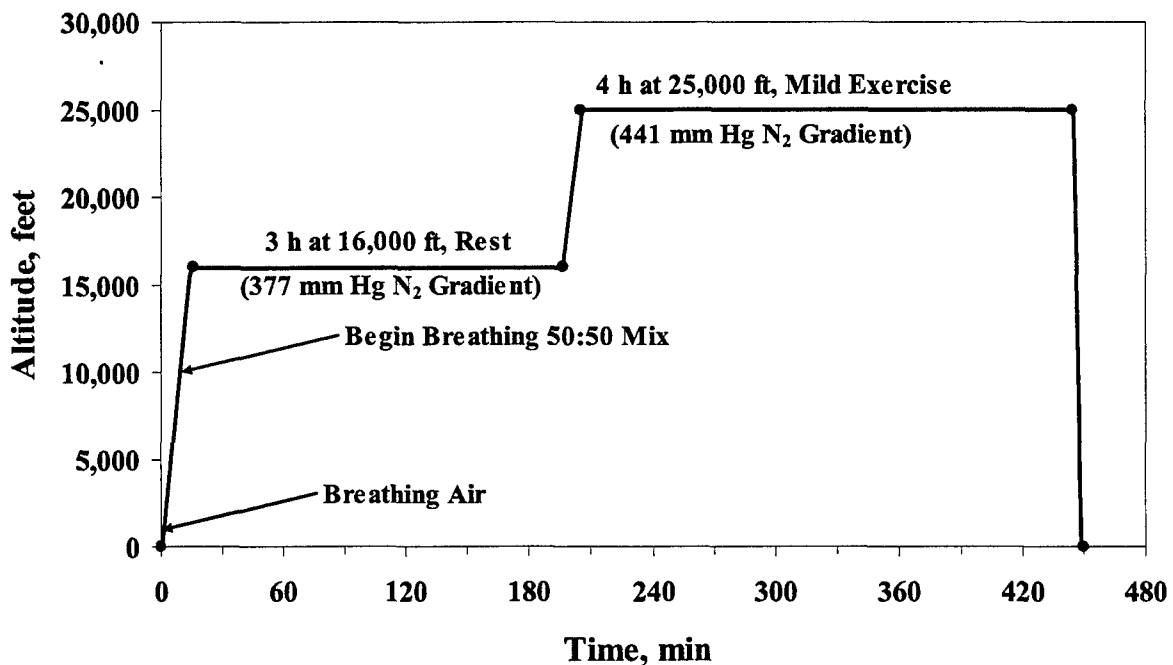


Figure 3. Test E exposure profile.

## RESULTS

### *Phase I:*

At the end of the 4-h exposures to 18,000 ft with zero prebreathe and mild exercise and breathing 60%N<sub>2</sub>/40%O<sub>2</sub> (Test A), the cumulative DCS incidence was 7% (Table III). The DCS incidence with the control exposures breathing 100% O<sub>2</sub> was 0%. This difference was not significant. However, there was a significant difference between the VGE incidence of Test A and the control values (70% versus 30%). Figure 4 shows the Test A cumulative onset curves

for DCS and VGE. Here again, the difference between the test and the control VGE values is clear. The Test B profile was identical to that of Test A except that it had heavy exercise instead of mild exercise. The cumulative DCS incidence at the end of the Test B exposures was 7% for both the test and the control exposures (Table III). Unlike Test A, however, there was no significant difference between the test and control cumulative VGE incidence in Test B (Figure 5).

**Table III: DCS and VGE results (n=40 in each test) for Tests A and B**

	Test A	Test B
<b><u>DCS</u></b>		
AFRL DCS Database (Controls)	0%	7%
DCS Incidence	7%	7%
Chi Sq	0.20	0.23
P	0.66	0.63
<b><u>VGE</u></b>		
AFRL DCS Database (Controls)	30%	63%
VGE Incidence	70%	69%
Chi Sq	7.73	0.21
P	0.01*	0.65

\*=significant

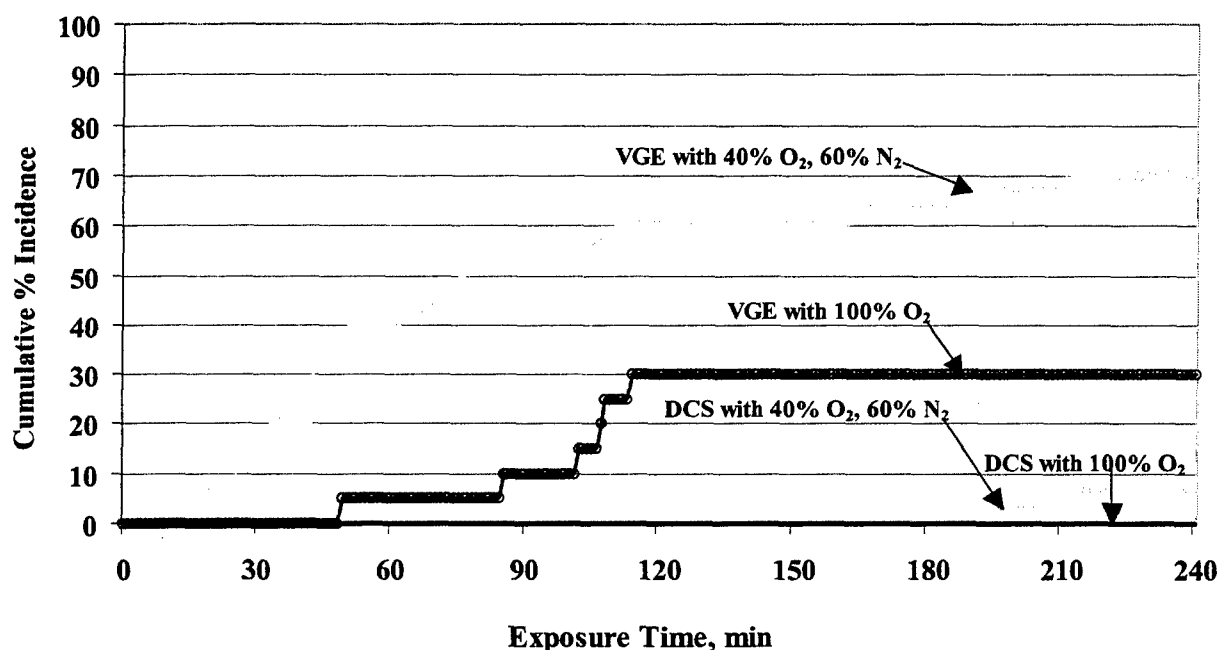


Figure 4. Cumulative % incidence of DCS and VGE at 18,000 ft; mild exercise; 240-min exposure; no preoxygenation (Test A)

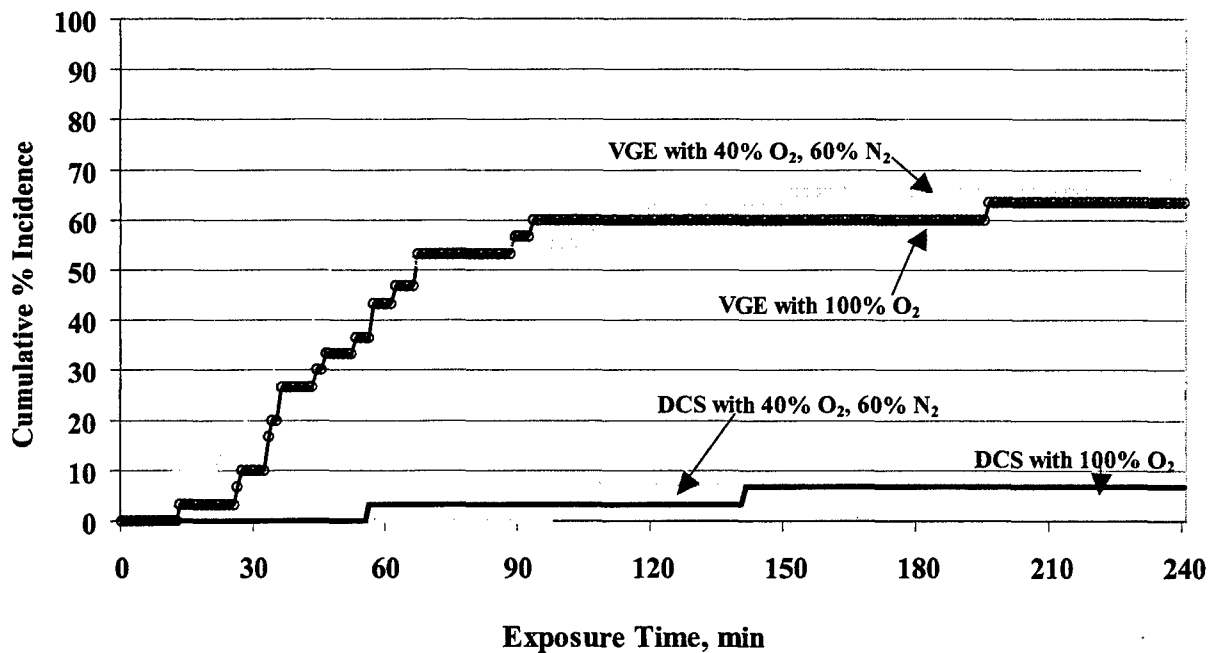


Figure 5. Cumulative % incidence of DCS and VGE at 18,000 ft; heavy exercise; 240-min exposure; no preoxygenation (Test B)

#### Phase II:

Results from Test C (at 22,500 ft breathing a mixture of 40%N<sub>2</sub>/60%O<sub>2</sub>, with zero prebreathe and mild exercise) showed that there was no significant difference between the test and the control in cumulative DCS incidence, but there was a very significant difference in the cumulative VGE incidence (Table IV, Figure 6). Table IV also contains the results from Test D (at 25,000 ft breathing an OBOGS mixture, with 90 min of prebreathe). Since there was no control available in the AFRL DCS Database for Test D, the only control value available was obtained from the ADRAC model. However, statistical comparison is not possible when the control value is generated by a predictive model. Since ADRAC does not predict VGE values,

there was no control for the VGE incidence. Figure 7 shows the cumulative DCS and VGE onset curves for Test D. The dotted line represents the DCS risk predicted by ADRAC for this exposure profile.

*Phase III:*

The DCS and VGE results for Tests E and F are shown in Table V and Figures 8 and 9. The third curve in these two figures represents the DCS and VGE results from the AFRL DCS Database from a previous study with the same exposure profile except that there was no stage denitrogenation and zero prebreathe (ZPB).

**Table IV: DCS and VGE results (n=40 in each test)**

	<u>Test C</u>	<u>Test D</u>
<b><u>DCS</u></b>		
AFRL DCS Database (Controls)	53%	31% (ADRAC)
DCS Incidence	43%	25%
Chi Sq	0.80	-
P	0.37	-
<b><u>VGE</u></b>		
AFRL DCS Database (Controls)	63%	N/A
VGE Incidence	90%	50%
Chi Sq	6.90	-
P	0.01*	-

\*=significant

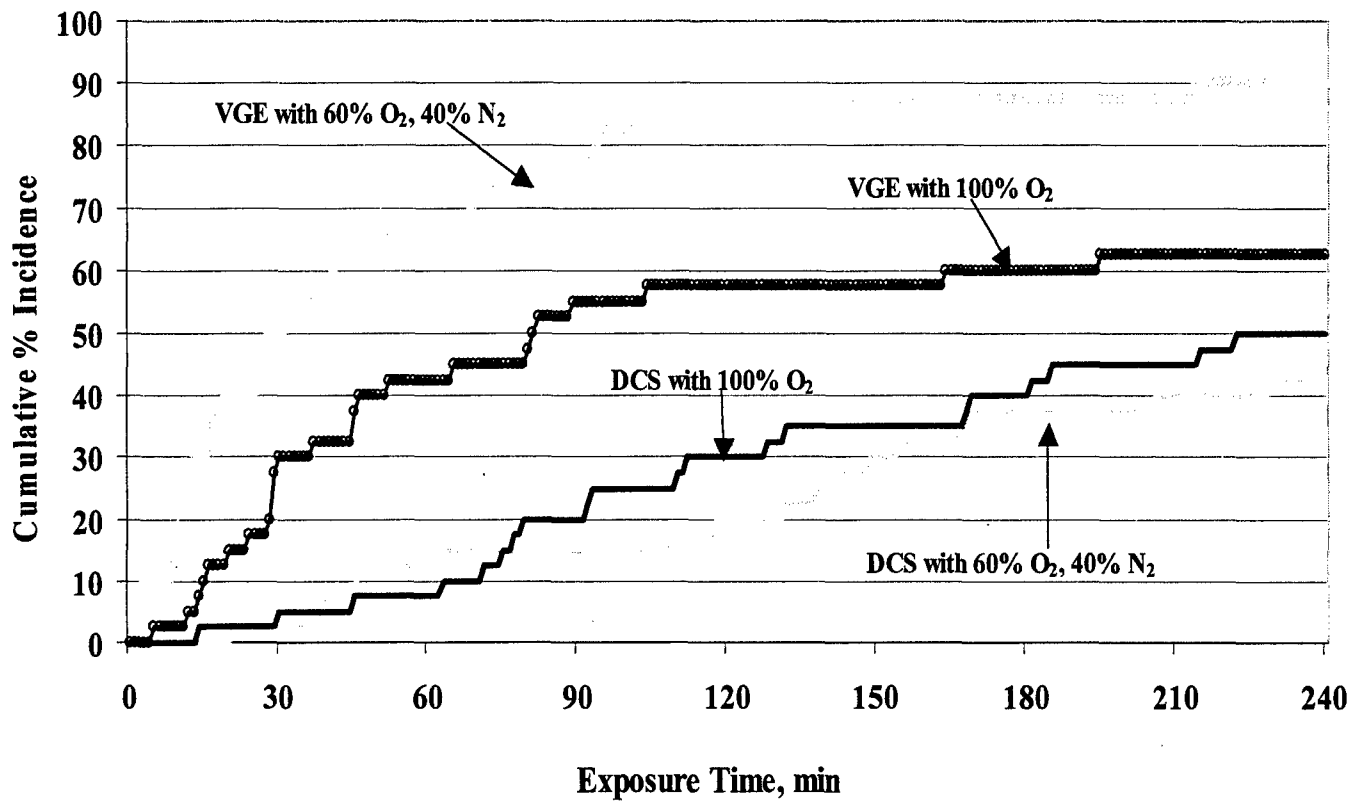


Figure 6. Cumulative % incidence of DCS and VGE at 22,500 ft; mild exercise; 240-min exposure; no preoxygenation, 40% N<sub>2</sub>/60% O<sub>2</sub> (Test C)

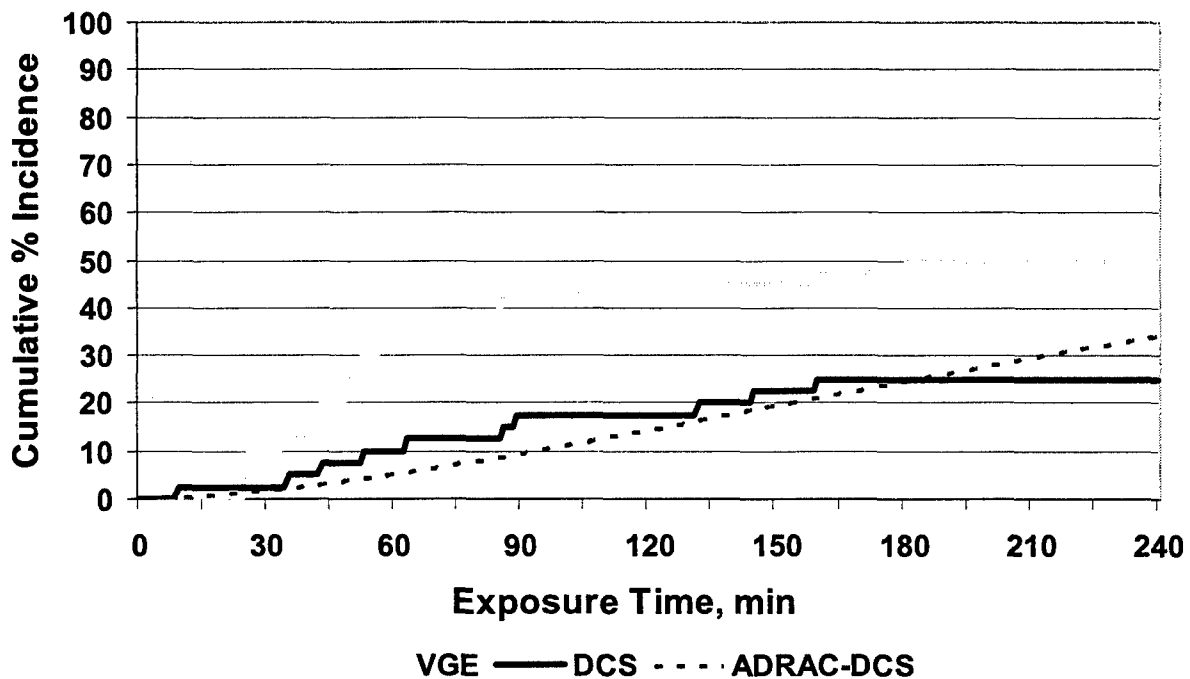


Figure 7. Cumulative % incidence of DCS and VGE at 25,000 ft; mild exercise; 240-min exposure; 90 min preoxygenation, 2.8%N<sub>2</sub>/4.2%Ar/93%O<sub>2</sub> (Test D)

Table V: DCS and VGE results (n=40 in each test) (Tests E and F)

	Test E	Test F
<b>DCS</b>		
DCS Incidence	50%	33 %
Chi Sq		2.53
P		0.11
<b>VGE</b>		
VGE Incidence	73%	48%
Chi Sq		7.94
P		0.005*

\*significance between Tests E & F

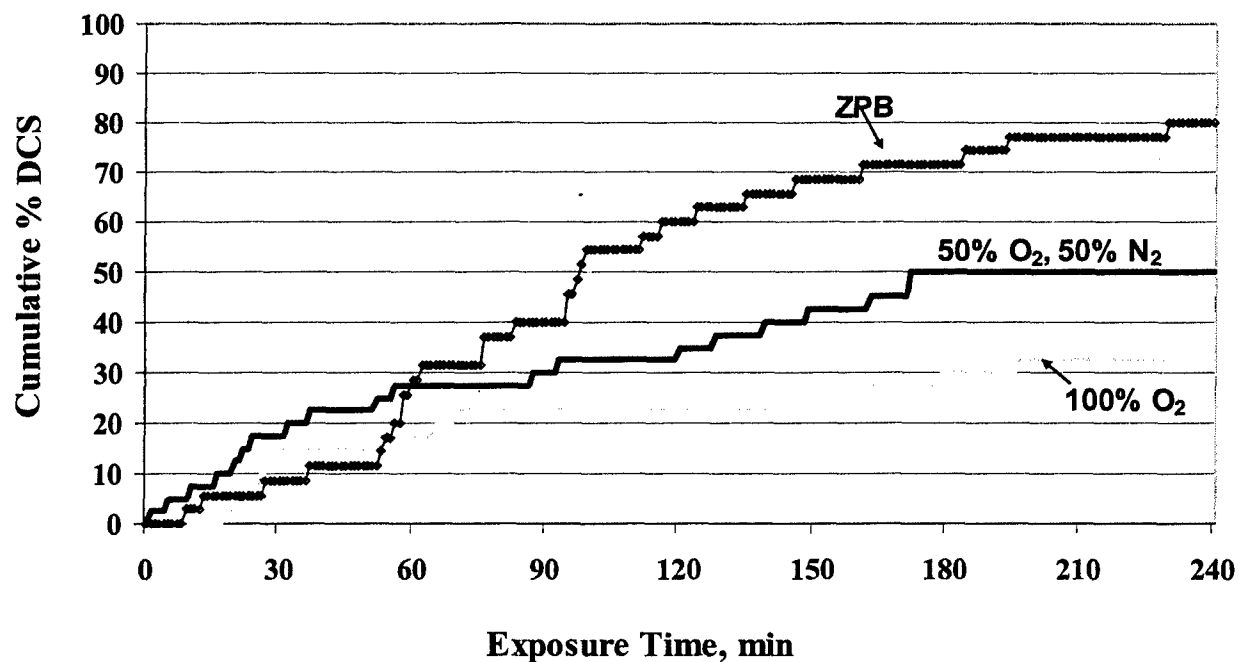


Figure 8. DCS symptom onset curves for Tests E and F compared to zero prebreathe (ZPB) exposures



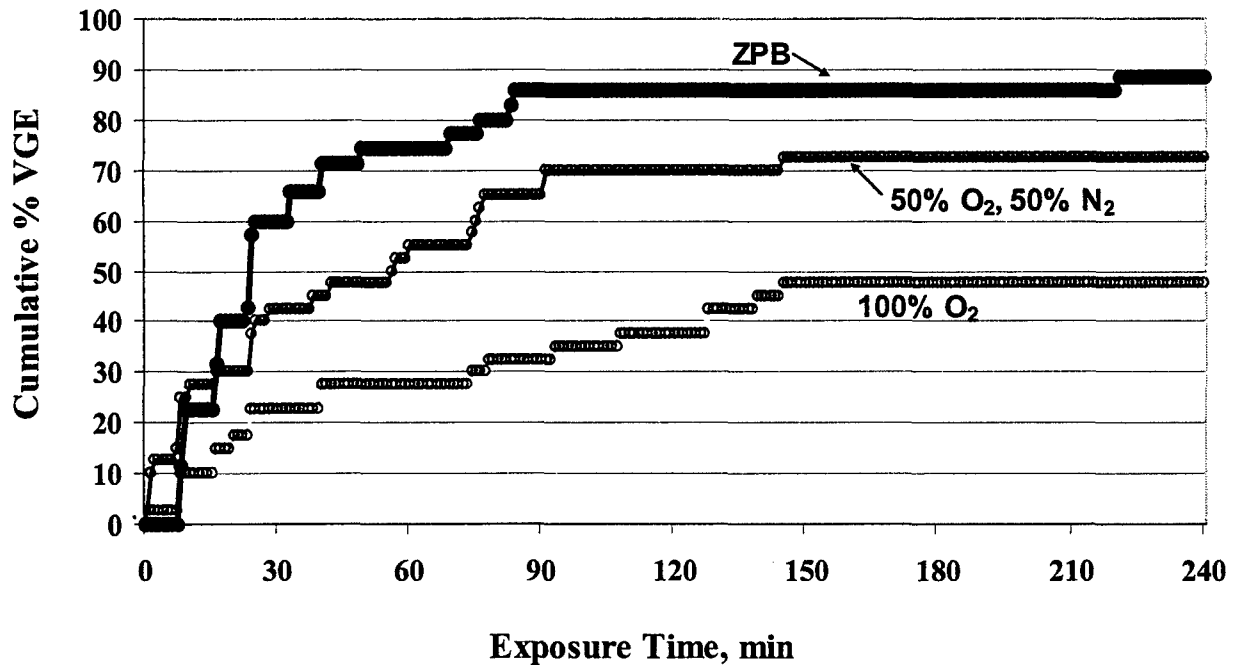


Figure 9. VGE onset curves for Tests E and F compared to zero prebreathe (ZPB) exposures

## DISCUSSION

Percentage is the most common way of referring to the components of a breathing gas mixture that an aviator is receiving. However, for defining DCS risk, the results of this study indicate that percentage of N<sub>2</sub> is not the most useful term. The total pressure of a breathing gas mixture is made up of the partial pressure components of that mixture (O<sub>2</sub>, N<sub>2</sub>, Ar). In the aerospace setting, if the percentage of each of those individual gases stays constant as altitude increases (pressure decreases), the partial pressures of each of those gases will obviously decrease. Assuming that with exposure to altitude the partial pressure of N<sub>2</sub> in the "slow" tissues (minimally perfused tissues slow to give up N<sub>2</sub>) in the body remains at ground-level saturation concentration, the PN<sub>2</sub> gradient will increase (the difference between the partial pressure of N<sub>2</sub> in

the tissues and the partial pressure of  $N_2$  in the ambient breathing gas). The resulting increased driving force for  $N_2$  to diffuse out of the body will decrease the tissue partial pressure of  $N_2$ , decrease the potential for bubble formation, and decrease the risk of DCS.

The results of Tests A, B, C, and D showed that the DCS risk at 18,000 ft, 22,500 ft, and 25,000 ft is NOT increased by breathing gas mixtures containing up to 60%  $N_2$  when compared to breathing 100%  $O_2$ . In Tests A and B at 18,000 ft, the DCS incidence was low, as expected, and it could be said that at such low DCS levels a statistical difference would be hard to find.

However, Test C at 22,500 ft showed a much higher DCS incidence than the control, but it was also not significant. Since the  $N_2$  and Ar percentages were very low, and the altitude was even higher, it is not surprising that there was no significant difference in DCS when compared with the use of 100%  $O_2$ . Lee and Hay (7), in a similar study, found that there were no significant differences in DCS between breathing 100% oxygen and breathing a mixture of 63%

oxygen/balance nitrogen. Their subjects were exposed to 25,000 ft for 4 hours at rest after one hour of 100% oxygen prebreathing at ground level. While the DCS risk was not different with high levels of  $N_2$ , the VGE incidence results showed significant differences in Tests A and C.

However, in Test B, with strenuous exercise, the VGE incidence was not different from the control. Significant VGE differences between high  $N_2$  breathing mixtures and 100%  $O_2$  (Figure 4) agree with our previous research (18) and with the results of Lee and Hay (7). A similar difference in VGE incidence was found when comparing results of Tests E and F (Figure 9). The VGE results in Test B (Figure 5) are contrary to these findings and are difficult to explain. It appears that no matter which gas was used, the heavy exercise results in high levels of VGE. Operationally, since VGE results are not good predictors of DCS symptoms (2, 10, 11), these VGE results have little application. However, the VGE results do suggest that there may be

some physiological decompression stress increase associated with the higher levels of  $N_2$  in the breathing gas at altitudes above 18,000 ft.

The Phase III Tests E and F indicate that breathing a 50% $N_2$ /50% $O_2$  mixture at 16,000 ft does not result in a significantly higher incidence of either DCS or VGE when compared to the same exposure breathing 100%  $O_2$ . However, it is clear from Figures 8 and 9 that the statistical significance is marginal. Thus, it appears that the concept discussed above (i.e., high levels of  $N_2$  in the breathing gas at altitude do not result in increased DCS) occurs only at altitudes higher than 16,000 ft. It is also clear from Figures 8 and 9 that there is significant benefit in in-flight denitrogenation using either breathing gas when compared to the same exposure with zero prebreathing (ZPB). The ZPB results were not from the present study, but were taken from previous research in the AFRL DCS Database.

The results of this research do not support the widely held view that high percentages of  $N_2$  are likely to increase DCS risk when breathed at altitude. These results support our hypothesis that it is the  $PN_2$  gradient increasing with altitude that determines the level of DCS risk, rather than the percentage of  $N_2$  per se. This effect is illustrated in Figure 10.

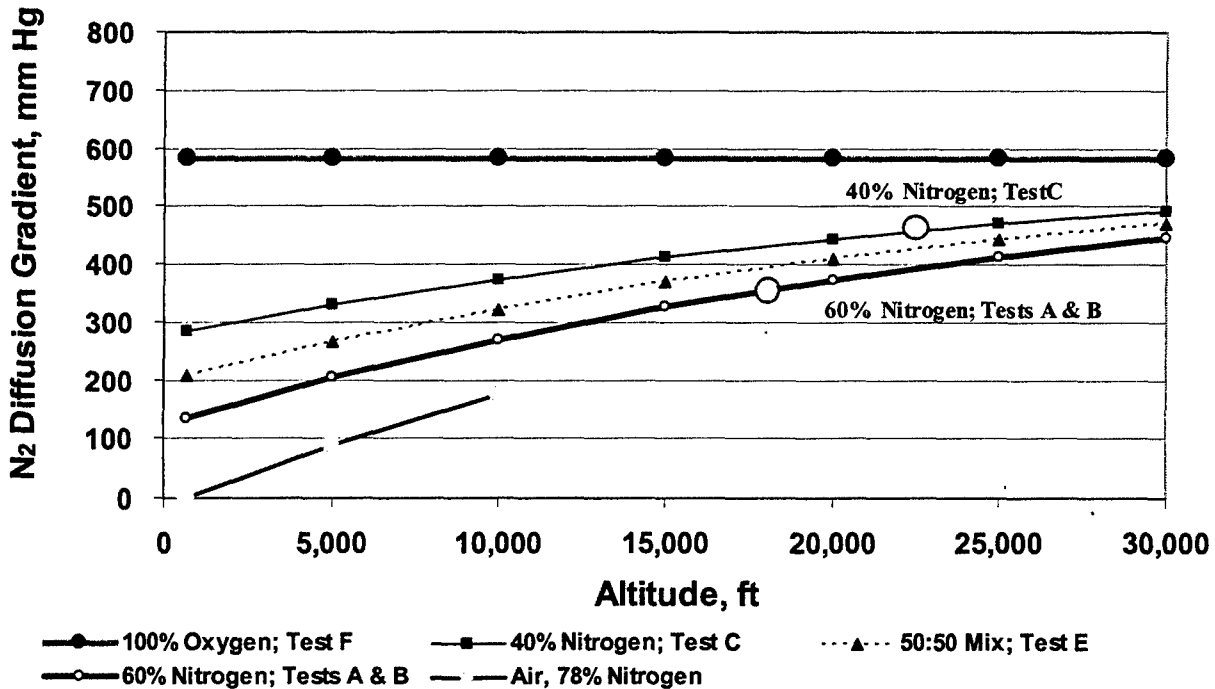


Figure 10. Calculated PN<sub>2</sub> Gradient vs Altitude

At ground level, breathing air, there is no PN<sub>2</sub> gradient. The tissues of the body are saturated with ground-level N<sub>2</sub> and are at equilibrium. As an individual ascends in altitude and continues to breathe air, the gradient increases and denitrogenation begins. The plot stops at 10,000 ft because, as a practical matter, the USAF requires the use of supplemental oxygen above 10,000 ft for hypoxia protection. The other extreme of the gradient spectrum is when 100% oxygen is the breathing gas and the gradient is at a maximum. This maximum does not change with altitude. It cannot get higher than a 583 mm Hg differential. Thus, denitrogenation is at its maximum when breathing 100% O<sub>2</sub>, whether prebreathing at ground level or flying at 20,000 ft. A family of curves for the PN<sub>2</sub> gradient and altitude can be plotted for the various breathing gas mixtures between 21% and 100% O<sub>2</sub>.

In Figure 10, the curves for the gas mixtures in Profiles A, B, C, E, and F are represented. It is clear from this figure that even though the breathing mixture contained 40% or 60% N<sub>2</sub>, the denitrogenation was relatively high when at 18,000 and 22,500 ft. It appears that the denitrogenation was sufficiently efficient compared to denitrogenation with 100% O<sub>2</sub> that the DCS risk was not significantly different. This concept permeates through all three phases of this study and explains the results, which are, seemingly, contradictory to conventional wisdom.

**Phase I: Is there an increased risk of altitude DCS resulting from the use of the NORMAL setting on the Narrow Panel Regulators of the AC-130 SPECTRE Gunships when compared to the 100% oxygen setting? If so, how much greater is that risk?**

The DCS incidence results of Tests A and B both show that there was no significant difference between the use of 60% nitrogen in the breathing gas and 100% oxygen (Table III, Figures 4 and 5). Thus, these data suggest that the use of the NORMAL setting on the AC-130 Narrow Panel Regulators would not result in a greater risk of DCS than using the 100% O<sub>2</sub> setting when operating at 18,000 ft. By use of the NORMAL setting, the aircraft oxygen supply would last longer and additional oxygen may not be required for extended flight times.

**Phase II: Will high levels of nitrogen in the breathing gas mixture produced by the On Board Oxygen Generating System (OBOGS) of the new AFSOC CV-22 Osprey result in an increased risk of DCS when compared to breathing 100% oxygen? If so, how much higher will that risk be?**

As with the results of Test A and B, Test C results show that a high level of nitrogen (40%) in the breathing gas does not result in an increased DCS risk (Table IV, Figure 6). The CV-22 OBOGS can produce breathing gas with as much as 40% N<sub>2</sub> at 22,500 ft. The results indicate that, under the conditions of this test, DCS symptom risk would not increase when compared to breathing 100% O<sub>2</sub>. Thus, as long as the OBOGS oxygen level is enough to prevent

hypoxia, DCS risk will not be significantly higher than if using 100% O<sub>2</sub> from liquid oxygen or high pressure cylinders. It becomes obvious that Test D was superfluous since, if 40 % N<sub>2</sub> does not increase DCS risk, then 2.8% N<sub>2</sub> certainly will not. There were no control exposures in the database with which to compare the results of Test D, so the ADRAC model was used to predict the DCS risk. No statistics are possible when comparing to the results of a model prediction, but the results appear similar. Both Tests C and D, and the Lee and Hay study (7), indicate that the CV-22 OBOGS product gas when used above 18,000 ft will not impact DCS risk.

**Phase III: The CV-22 is expected to operate unpressurized up to altitudes of 25,000 ft for long periods of time. A very high risk of DCS is predicted for such operations. Assuming the use of ground-level preoxygenation (prebreathing) for reducing DCS risk is impractical for AFSOC, how effective would in-flight (stage) denitrogenation by breathing 100% or OBOGS product gas be for reducing DCS risk?**

The results from Tests E and F show that stage denitrogenation, even with 50% N<sub>2</sub> in the breathing gas, will significantly reduce the risk of DCS. The zero-prebreathe DCS risk for 4-hour exposures to 25,000 ft can be seen in Figures 8 and 9. Breathing 100% O<sub>2</sub> for 3 hours at 16,000 ft prior to ascent to 25,000 ft for 4 hours reduced the DCS risk to 33% (Table III, Figure 8) from 80% with zero prebreathe. Using a 50:50 mix under the same conditions reduced the DCS risk to 50% (Table III, Figure 8). Thus, in-flight "prebreathing" would be of value in reducing DCS risk if operationally feasible, and would significantly reduce DCS risk compared to zero prebreathe flights.

In Phases I and II there were no significant differences between the DCS risk when breathing 100% O<sub>2</sub> and two different N<sub>2</sub>O<sub>2</sub> mixes. In Phase III, however, there was a marginal difference between breathing the 50:50 mix and the 100% O<sub>2</sub> gas in the DCS risk (50% vs 33%).

Most likely this was due to the lower altitude at which the gas was breathed. Referring to Figure 10, it appears that a breathing gas high in  $N_2$  begins to have an effect between 16,000 and 18,000 ft. Thus, at altitudes less than 16,000 ft, the use of 100%  $O_2$  for denitrogenation is recommended.

## CONCLUSION

The nitrogen partial pressure gradient partly determines the extent and rate of denitrogenation during altitude exposure. The degree of denitrogenation, in turn, determines the potential for bubble formation and DCS incidence. Within the parameters of the experiments of this study, it is concluded that the increased nitrogen levels in the breathing gas while at altitudes of 18,000 to 25,000 ft did not significantly increase DCS risk. Although it is likely that there is greater denitrogenation when using 100%  $O_2$  versus using a high  $N_2$  mix at these altitudes, as reflected in the increased VGE incidence with high  $N_2$  breathing gases, apparently the magnitude of that difference is not enough to impact the risk of clinical DCS.

Stage "prebreathing," or in-flight denitrogenation, at 16,000 ft prior to ascent to 25,000 ft is effective in reducing the DCS risk when compared to zero prebreathe exposures. It appears that 16,000 ft is a denitrogenation boundary altitude for use of high levels of  $N_2$  in the breathing gas, and 100%  $O_2$  is recommended at altitudes below 16,000 ft when denitrogenation (prebreathing) is required.

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